Nutrition Column

Nutrition and HIV-Positive Pregnancy

Kristen S. Montgomery, PhD, RN

KRISTEN MONTGOMERY is a postdoctoral research fellow in the School of Nursing at the University of Michigan in Ann Arbor, Michigan.

Abstract

When an HIV-positive woman becomes pregnant, additional nutritional considerations are warranted. Compared to routine prenatal nutritional assessment and intervention, pregnant HIV-positive women have increased needs to promote a healthy outcome. This column contains information on HIV and pregnancy, nutrition and infection, and nutrition for HIV-positive pregnancy. This content can be integrated into childbirth education settings to improve care to women who are HIV-positive.

***Journal of Perinatal Education,* 12(1), 42–47;** *nutrition, HIV-positive pregnancy.*

Pregnancy is a time of increased nutritional need. The need for both absolute calories and some specific nutrients are augmented during this period. Satisfying additional nutritional needs contributes to both fetal development and maternal stores for labor, delivery, and breastfeeding. However, when pregnancy is complicated by human immunodeficiency virus (HIV) infection, specific additional considerations are warranted. See Box for background on HIV infections.

HIV and Pregnancy

Pregnancy complicated by HIV infection is considered high-risk (Jones & Verdejo, 1999). A normal pregnant woman is slightly immunosuppressed to protect the fetus from a maternal antibody response to genetic material that is foreign (e.g., the father's genes). The pregnant woman is not more susceptible to infection, but when an infection does occur, it is much more difficult to treat (Gilbert & Harmon, 1998).

While fertility is decreased following HIV infection,

Background on HIV Infection

Normal immune function includes specific and nonspecific responses to invading foreign substances (e.g., bacteria and viruses) (Jones & Verdejo, 1999). Some capacity for "remembering" foreign substances exists, allowing for more rapid response and irradication in certain circumstances. The immune system also serves to maintain a balance among bacteria that are considered normal flora in or on the body—for example, the normal bacteria flora of the gastrointestinal tract or vagina.

HIV infection results in an alteration of the genetic code of susceptive cells (Jones & Verdejo, 1999). After infection, the resulting provirus may remain inactive for a considerable amount of time. This inactivity contributes to the variation seen in latency to the disease state. The trigger for active replication after a period of dorminancy is currently unknown (Jones & Verdejo, 1999).

After viral replication begins, the host cell dies. Over a period of time, CD4+ cells (host cells) become so few in number that immune function is diminished. Diminished immune function permits normal body flora to proliferate, which can result in opportunistic infections (e.g., candida [yeast] infection).

The HIV-1 virus causes the majority of HIV infections; however, an HIV-2 virus also exists. HIV infection is transmitted through contaminated needles, sexual contact with an infected partner, breastfeeding, pregnancy/labor/delivery, and blood/blood product transfusions (Jones & Verdejo, 1999). Infection among women most commonly occurs through heterosexual contact with an infected partner. Severity of infection is monitored through serial CD4 levels. CD4 levels and viral loads are used to guide treatment and prophylaxis measures, as well as the efficacy of treatment regimes.

Initial HIV infection generally results in a decline in CD4+T lymphocytes and a prompt increase in the amount of virus present in the blood (viral load) (Jones & Verdejo, 1999). An acute illness may also manifest as the virus spreads throughout the body. The viral load in the blood begins to decrease after the body begins a response to fight it (Jones & Verdejo, 1999). This decrease represents a period of clinical latency; however, the virus continues to replicate and the progressive decline of CD4+T lymphocytes continues. This period can last for 10 years or more. At the end of this period, the virus can no longer be contained and an increase in viral load is again observed (Jones & Verdejo, 1999). This change is often accompanied by an acquired immune deficiency syndrome (AIDS) defining illness such as Kaposi's sarcoma.

pregnancy does occur (Glynn et al., 2000). Pregnancy does not worsen HIV/AIDS status (Alliegro et al., 1997), and HIV/AIDS does not worsen pregnancy outcomes other than transmission (American Medical Association, 1997; Sweet & Gibbs, 1995). Most cases of perinatal transmission occur late in gestation or through breastfeeding (Jones & Verdejo, 1999).

Nutrition and Infection

Nutritional status is easily compromised during any type of infection. Generalized infections often result in reduced food intake and absorption of nutrients (Friis, 2002). Utilization and loss of nutrients are also increased during an acute infection. Additional characteristics of the infection (e.g., fever, mouth sores, and fatigue) can further contribute to poor nutrition. Medications used to treat an underlying infection can further contribute to poor nutritional status when they have side effects such as nausea, vomiting, and dry mouth or they alter taste or smell.

Nutritional status can also influence infection. Iron deficiency has been associated with soil eating in developing countries, and low intake of zinc has been shown to reduce spontaneous physical activity (Friis, 2002).

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Supplemental iron during latent or uncontrolled infections may be more beneficial to the infecting organism than the infected individual. Iron can contribute to the growth and replication of infectious agents (Friis, 2002). Zinc deficiency has been associated with HIV infection and results in a reduction in the number of circulating T lymphocytes (Kupka & Fawzi, 2002). Host immunity plays a major role in how behavior may influence disease (Friis, 2002).

Nutrition for HIV-Positive Individuals

Multiple micronutrient deficiencies develop early in the course of HIV infection and contribute to increased micronutrient needs among HIV-infected individuals (Friis, Gomo, & Michaelsen, 2002). Poor absorption, reduced intake, and increased nutrient utilization and loss also contribute to the development of nutrient deficiencies. Individuals infected with HIV, however, tend to accumulate iron in tissues, especially bone marrow, the brain, muscle, liver, and spleen (Weinberg, G. A., Boelaert, & Weinberg, E. D., 2002). This iron accumulation or loading is believed to be related to a chronic inflammatory response that involves the retention of iron (Boelaert, Weinberg, G. A., & Weinberg, E. D., 1996; Jurado, 1997). Iron may also accumulate with repeated transfusion of packed red blood cells (Boelaert et al., 1996) or by smoking tobacco (Mateos, Brock, & Perez-Arellano, 1998). While not conclusive, preliminary research efforts indicate that larger iron stores result in a faster progression of HIV infection (Weinberg et al., 2002).

Low levels of B_{12} may be associated with progression to AIDS (Tang, Graham, Chandra, & Saah, 1997), impaired cognitive function, and drug toxicity (Tang & Smit, 1998). High levels of some B vitamins have been associated with increased survival (Kanter et al., 1999; Tang, Graham, & Saah, 1996). Inadequate nutritional intake or disturbance in the body's ability to process nutrients can result in the loss of lean body mass and in wasting (Winson, 2001). Death occurs when a person's weight reaches about 60% of his/her ideal body weight, regardless of the cause (Kotler, Tierney, Wang, & Pierson, 1989). Severity of wasting can be assessed through body weight, body mass index, triceps skinfold thickness, mid-upper arm circumference, functional power, and laboratory testing (Winson, 2001).

To date, few authors or associations have made nutrition recommendations for HIV-positive individuals. Buy and Hussey (2002) recommend that individuals mildly infected with HIV or on antiretroviral therapy receive general vitamin supplementation at one recommended dietary allowance (RDA) per day. Those with severe infection should be supplemented with multivitamins for at least two RDAs. Nutrition counseling has been found to improve energy intake and certain aspects of cognitive function in HIV-positive individuals (Rabeneck, et al., 1998). Risk of lean body mass loss can be improved when nutrition counseling is combined with nutritional interventions (Stack, Bell, Burke, & Forse, 1996).

Nutrition for HIV-Positive Pregnancy

In animal models, nutrient deficiencies during pregnancy have been shown to affect the immune function of the next generation (Friis, 2002). In HIV-negative humans, individuals born during the hunger season (and more likely to have nutritional deficiency during pregnancy) in Gambia had higher rates of general infectious disease in adulthood when compared to individuals born during the harvest season (Friis, 2002). In this instance, it is possible that the immune function of a nutrient-deficient fetus aged prematurely (immunosenescence) or reached its peak immune response related to the nutrient deprivation incurred during fetal life. Thus, it seems the immune response mechanisms did not have adequate nutrients to develop normally and perhaps developed more prematurely.

Vitamin A

Vitamin A is essential for reproduction (Semba, 2002). Male animals that are deficient in vitamin A are not as able to produce sperm. Low vitamin-A levels are also hypothesized to negatively affect female fertility. Vitamin-A deficiency has also been associated with placental infections. Pregnancy increases the risk of vitamin-A deficiency for both the mother and newborn, and vitamin-

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A deficiency in the HIV-positive, pregnant mother has been associated with increased infant mortality (Semba et al., 1998). Recent research has not shown the benefit of vitamin-A supplementation during pregnancy on infant mortality (Katz et al., 2000). However, maternal supplementation with carotene recently showed a 50% reduction in maternal morbidity (West et al., 1999). Lower circulating levels of carotenoid concentrations have been found in pregnant women with pre-eclampsia in the U.S. and Nigeria, and low circulating carotene concentrations have been found in pregnant women with preterm rupture of the fetal membranes (Semba, 2002).

Iron

Most women in the U.S. are advised to take supplemental iron during pregnancy regardless of evidence of iron deficiency (Weinberg et al., 2002). For uncomplicated pregnancies, this routine practice is thought to be safe and, in certain circumstances, may have some benefit (Weinberg et al., 2002). Few data are available on how HIV/AIDS influences the pregnant woman's iron status; however, Friis and colleagues (2001) reported that HIV infection alters several micronutrients in pregnant women living in Zimbabwe. When compared to noninfected pregnant women, HIV-positive, pregnant women had lower concentrations of serum folate, ferritin, and hemoglobin (Friis et al., 2001). Iron supplementation for most pregnant women in developing countries is probably safe because correlations have not been found between iron status and markers of HIV disease severity (Semba et al., 2001). However, iron overload may be harmful to some individuals infected with HIV. Additional iron provides nutrients that supplement viral replication, and the iron status of an individual plays a role in the development of opportunistic infections (Weinberg et al., 2002). Excessive iron consumption can be avoided with decreased intake of red meat and alcohol (which facilitates iron absorption), reduction or elimination of iron supplementation, careful monitoring of processed foods that may have been iron fortified (e.g., loaf breads), reduction of unnecessary blood transfusions, and an increased intake of iron-chelating plant foods (e.g., soy products, cereals, and teas containing phytic acid, tannins, and polyphenols), which may decrease gastrointestinal absorption of iron (Weinberg et al., 2002). Reduced exposure to tobacco smoke, asbestos fibers, and urban air particulates is also helpful in reducing iron overload.

Vitamin Supplementation

Pregnant, HIV-positive women should be encouraged to obtain needed nutrients from a balanced diet. At this time, no specific RDAs are available for pregnant, HIV-positive women. A prenatal multivitamin or micronutrient supplementation is likely to be beneficial and is an easy, cost-effective means to improve maternal and neonatal health. Vitamin supplementation should occur for as much of pregnancy as possible and should continue for three months postpartum.

While several nutrients have been identified as possibly beneficial to pregnant, HIV-positive women, it is not practical to completely tailor micronutrient supplementation in most instances. Thus, investigators have examined multivitamin supplementation. Multivitamin and vitamin-A supplementation have not influenced rates of maternal-to-child transmission; however, benefits of supplementation did include improvement of CD4 lymphocyte counts and infant birth weight, as well as a reduced risk of growth restriction and severe prematurity (Fawzi et al., 2000). Research is also needed to determine if zinc plays a role in perinatal transmission or poor birth outcomes (Kupka & Fawzi, 2002). Supplementation also improves anemia and maternal mortality and reduces the risk of some congenital anomalies and fetal mortality (Huffman, Baker, Shumann, & Zehner, 1999). When combined with vitamins C and E, high doses of the B vitamins improved pregnancy outcome and increased CD4 counts in one sample of HIV-positive, pregnant women (Fawzi et al., 2000). Adequate protein intake is essential to maintain cell-mediated immunity, complement, and phagocytes (Gilbert & Harmon, 1998).

Food Handling and Eating Patterns

Pregnant, HIV-positive women can be particularly vulnerable to food-borne infections (Whitney & Rolfes, 2002). Guidelines for handling, storing, and cooking foods should be reinforced, and raw or undercooked

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seafood should be avoided. Pregnant women who experience gastrointestinal upset from medications associated with HIV treatment can be encouraged to eat small, frequent meals throughout the day, consuming liquids between meals instead of with meals and snacking on crackers (Sherman, D., & Sherman, N., 2001).

Summary

Perinatal education for pregnant women with HIV warrants a special session on nutrition as early in the pregnancy as feasible. Continued monitoring and counseling throughout the pregnancy may be helpful in providing the infant with the best start possible. Additional general information on HIV and pregnancy is available in the article titled "Resource Column: HIV and Pregnancy Web Sites," which was published in a previous issue of this journal (Montgomery, 2002).

References

Alliegro, M. B., Dorrucci, M., Phillips, A. N., Pezzotti, P., Boros, S., Zaccarelli, M., Pristera, R., & Rezza, G. (1997). Incidence and consequences of pregnancy in women with known duration of HIV infection. *Archives of Internal Medicine, 157*, 2585–2888.

American Medical Association. (1997). *Treatment center: Perinatal transmission.* Retrieved August 2002 from http://www.ama-assn.org/aids

Boelaert, J. R., Weinberg, G. A., & Weinberg, E. D. (1996). Altered iron metabolism in HIV infection: Mechanisms, possible consequences, and proposals for management. *Infectious Agents of Disease, 5,* 36–46.

Buy, H., & Hussy, G. (2002). Micronutrients in the case management of HIV infection. In H. Friis (Ed.), *Micronutrients and HIV Infection* (pp. 201–217). Boca Raton, FL: CRC Press.

Fawzi, W., Msamanga, G., Hunter, D., Urassa, E., Renjifo, B., Mwakagile, D., Hertzmark, E., Coley, J., Garland, M., Kapiga, S., Antelman, G., Essex, M., & Spiegelman, D. (2000). Randomised trial of vitamin supplements in relation to vertical transmission of HIV-1 in Tanzania. *Journal of AIDS, 23*, 246–254.

Friis, H. (2002). Micronutrients and HIV infection: An introduction. In H. Friis (Ed.), *Micronutrients and HIV Infection* (pp. 1–21). Boca Raton, FL: CRC Press.

Friis, H., Gomo, E., Koestel, P., Ndhlovu, P., Nyazema, N., Krarup, H., & Michaelsen, K. F. (2001). HIV and other predictors of serum folate, serum ferritin, and hemoglobin in pregnancy: A cross-sectional study in Zimbabwe. *American Journal of Clinical Nutrition, 73,* 1066–1073.

Friis, H., Gomo, E., & Michaelsen, K. F. (2002). Micronutrient interventions and the HIV pandemic. In H. Friis (Ed.), *Micronutrients and HIV Infection* (pp. 219–246). Boca Raton, FL: CRC Press.

Gilbert, E. S., & Harmon, J. S. (1998). Sexually and nonsexually transmitted genitourinary infections. In E. S. Gilbert & J. S. Harmon (Eds.), *Manual of High-Risk Pregnancy and Delivery* (2nd ed.) (pp. 618–652). St. Louis, MO: Mosby.

Glynn, J. R., Buve, A., Carael, M., Kahindo, M., Macauley, I. B., Musonda, R. M., Jungmann, E., Tembo, F., & Zekeng, L. (2000). Decreased fertility among HIV-1-infected women attending antenatal clinics in three African cities. *Journal of Acquired Immune Deficiency Syndrome, 25*, 345–352.

Huffman, S., Baker, J., Shumann, J., & Zehner, E. (1999). The case for promoting multiple vitamin and mineral supplements for women of reproductive age in developing countries. *Food and Nutrition Bulletin, 20,* 379–380.

Jones, M., & Verdejo, T. (1999). Human immune deficiency virus and acquired immune deficiency syndrome. In L. K. Mandeville & N. H. Troiano (Eds.), *AWHONN's High-Risk and Critical Care Intrapartum Nursing* (2nd ed.), (pp. 302–321). Philadelphia: J. B. Lippincott.

Jurado, R. L. (1997). Iron, infections, and anemia of inflammation. *Clinical Infectious Disease, 25,* 888–895.

Kanter, A. S., Spencer, D. C., Steinberg, M. H., Soltysik, R., Yarnold, P. R., & Graham, N. M. (1999). Supplemental vitamin B and progression to AIDS and death in black South African patients infected with HIV. *Journal of Acquired Immune Deficiency Syndrome, 21*(3), 252–253.

Katz, J., West, K. P., Khatry, S. K., Pradhan, E. K., LeClerq, S. C., Christian, P., Wu, L. S., Adhikari, R. K., Shrestha, S. R., & Sommer, A. (2000). Maternal low-dose vitamin A or carotene supplementation has no effect on fetal loss and early infant mortality: A randomized cluster trial in Nepal. *American Journal of Clinical Nutrition, 71,* 1570–1576.

Kotler, D. P., Tierney, A. R., Wang, J., & Pierson, R. N., Jr. (1989). The magnitude of body cell mass depletion in the timing of death from wasting in AIDS. *American Journal of Clinical Nutrition, 50,* 444–447.

Kupka, R., & Fawzi, W. (2002). Zinc nutrition and HIV infection. *Nutrition Review, 60(3),* 69–79.

Mateos, F., Brock, J. H., & Perez-Arellano, J. L. (1998). Iron metabolism in the lower respiratory tract. *Thorax, 53,* 594–600.

Montgomery, K. S. (2002). Web sites that address HIV and pregnancy. *Journal of Perinatal Education, 11*(4), 41–43.

Rabeneck, L., Palmer, A., Knowles, J., Seidehamel, R., Harris, C., Merkel, K., Risser, J., & Akrabawi, S. (1998). A randomized controlled trial evaluating nutrition counseling with or without oral supplementation in malnourished HIV-infected patients. *Journal of the American Dietetics Association, 98,* 434–438.

Semba, R. D. (2002). Vitamin A, carotenoids, and HIV infection. In H. Friis [Ed.], *Micronutrients and HIV infection* (pp. 73–90). Boca Raton, FL: CRC Press.

Semba, R. D., Miotti, P. G., Chiphangwi, J. D., Dallabetta, G., Yang, L. P., Saah, A., & Hoover, D. (1998). Maternal vitamin A deficiency and infant mortality in Malawi. *Journal of Tropical Pediatrics, 44*, 232–234.

Semba, R. D., Taha, T. E., Kumwenda, N., Mtimavalye, L., Brodhead, R., Micotti, P. G., & Chiphangwi, J. D. (2001). Iron status and indicators of human immunodeficiency virus disease severity among pregnant women in Malawi. *Clinics of Infectious Disease, 32*, 1496–1499.

Sherman, D., & Sherman, N. (2001). HIV/AIDS and pregnancy. In C. A. Kirton, D. Talotta, & K. Zwolski (Eds.), *Handbook of HIV/AIDS Nursing* (pp. 361–379). St. Louis, MO: Mosby.

Stack, J., Bell, S., Burke, P., & Forse, A. (1996). High-energy, high-protein, oral, liquid, nutrition supplementation in patients with HIV infection: Effect on weight status in relation to incidence of secondary infection. *Journal of the American Dietetic Association, 96,* 337–341.

Sweet, R. L., & Gibbs, R. S. (1995). *Infectious diseases of the female genital Tract* (3rd ed.). Baltimore: Williams & Wilkins.

Tang, A. M., Graham, N. M., Chandra, R. K., & Saah, A. J. (1997). Low serum vitamin B-12 concentrations are associated with faster human immunodeficiency virus type 1 (HIV-1) disease progression. *Journal of Nutrition, 127*(2), 345–351.

Tang, A. M., Graham, N. M., & Saah, A. J. (1996). Effects of micronutrient intake on survival in human immunodeficiency virus type 1 infection. *American Journal of Epidemiology, 14,* 1244–1256.

Tang, A. M., & Smit, E. (1998). Selected vitamins in HIV infection: A review. *AIDS Patient Care Standards, 12,* 263–273.

Weinberg, G. A., Boelaert, J. R., & Weinberg, E. D. (2002). Iron and HIV infection. In H. Friis (Ed.), *Micronutrients and HIV Infection* (pp. 135–180). Boca Raton, FL: CRC Press.

West, K. P., Katz, J., Khatry, S. K., LeClerq, S. C., Pradhan, E. K., Shrestha, S. R., Connor, P. B., Dali, S. M., Christian, P., Pokhrel, R. P., & Sommer, A. (1999). Double blind, cluster randomized trial of low dose supplementation with vitamin A or carotene or mortality related to pregnancy in Nepal. *British Medical Journal, 318,* 570–575.

Whitney, E. N., & Rolfes, S. R. (2002). *Understanding Nutrition* (9th ed.). Belmont, CA: Wadsworth.

Winson, G. (2001). HIV/AIDS nutritional management. In C. A. Kirton, D. Talotta, & K. Zwolski (Eds.), *Handbook of HIV/AIDS Nursing* (pp. 344–359). St. Louis, MO: Mosby.